one analysis of unprotected anal intercourse with primary partners, the sample size was 17 in the intervention community and 9 in the comparison community.

However, sample size may not be the only reason for statistical failure. Indeed, there are many other ways in which we set ourselves up for failure. Clearly, the effect size one can expect depends heavily upon the outcome measure selected. Given a human immunodeficiency virus (HIV) prevention intervention, if one were to evaluate the intervention by considering reductions in HIV incidence, one would have very different expectations of success than if one were to consider reductions in sexually transmitted disease (STD) rates, the number of unprotected sex acts, the proportion of times condoms were used, the proportion of people engaging in unprotected anal sex, or the proportion of people who always (or never) use condoms. To put this somewhat differently, is it reasonable to expect an intervention to have a medium effect size on all of these variables?

Jacob Cohen[3,4] did the field a great service by reminding us that our ability to detect significant differences depended upon sample size as well as natural variability, and by chastising us for not having large enough samples to detect meaningful differences.[3,4] Unfortunately, his formulas and tables for determining the sample size necessary to detect such a meaningful difference have been badly misused.[5] Many investigators simply calculate (or look up) the sample size necessary to detect a medium size effect, irrespective of the outcome variable or variables being considered. Since the likelihood of a medium or large effect often is unrealistic, this turns out to be a process that tends to generate findings of "no difference."

Perhaps the cardinal rule is that the outcome variable selected must be consistent with, and sensitive to, the purpose of the intervention. For example, in the Kegeles et al. study, the intervention was designed to increase the likelihood that the young men would engage in safer sex. As described above, Kegeles et al. evaluated their intervention by looking for a reduction in the proportion of men who engaged in *any* act of unprotected anal sex. The question that must be asked is whether this measure fully captures the effect of their intervention. Not reflected in this outcome measure would be a person who had reduced the number of unprotected acts of anal intercourse from 50 at baseline to 25 at follow-up, or one who went from no condom use to 75% condom use, or who reduced the number of acts of anal sex or the number of partners, or who substituted masturbation or "outercourse" for intercourse. Did the outcome measure selected by the authors ask too much of their intervention? Was it fair to view a person who reduced unprotected sex acts from 100 to 0 as no more a success than one who reduced such acts from 1 to none? So stringent an outcome measure surely lowered the probability of detecting a significant intervention effect.

Even assuming that the ultimate goal of a public health behavior change intervention is to reduce morbidity and mortality, the fact remains that behavior change interventions are, first and foremost, designed to change behaviors. Thus, the first step in the development of an intervention should be the identification of the behavior (or behaviors) one wishes to change. The choice of this behavior should be based not only on sound epidemiological evidence linking the behavior to a biomedical outcome of public health interest, but also on the prevalence (or mean value and variance) of this behavior in the population being studied. Although a complete discussion of the relations among behavioral and biological measures is beyond the scope of this editorial, it must be recognized that, in the area of HIV prevention, the impact of a given behavior change on either a sexually transmitted disease or HIV, like the impact of a reduction in sexually transmitted diseases on HIV, will depend on such things as the prevalence of the disease, the ease with which it is transmitted from an infected to an uninfected partner (which varies, among other things, with gender), the sexual mixing patterns in the population, and the periods during which the condition is most infectious. Thus, although it is essential to explore the impact of a behavior change on morbidity, the most appropriate outcome measure for evaluating a behavior change intervention is a measure of the behavior per se.

Clearly, if we are going to provide fair tests of the efficacy or effectiveness of community-based (and other) interventions, it will be necessary to be much more careful in our choice of outcome measure and in our expectations about the size of an effect that an intervention can have on that measure. Outcome measures must be sensitive to the purpose of the intervention, and when a small-sized effect is meaningful (and all we can expect) for a given outcome measure, we must make sure that we have the sample size necessary to detect such an effect. Until this is done, we will continue to find little statistical support for the effectiveness of community-based and other interventions. □

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## References

1. Kegeles SM, Hays RB, Coates TJ. The Mpowerment Project: a community-level HIV prevention intervention for young gay men. 1996;86:1129–1136.
2. Susser M. Editorial: the tribulations of trials—intervention in communities. *Am J Public Health.* 1995;85:156–158. Editorial.
3. Cohen J. The statistical power of abnormal-social psychological research: a review. *J Abnorm Soc Psychol.* 1962;65:145–153.
4. Cohen J. *Statistical Power Analysis for the Behavioral Sciences.* Hillsdale, NJ: Lawrence Erlbaum Associates; 1969.
5. Cohen J. The earth is round ($p < .05$). *Am Psychol.* 1994;49:997–1003.

# Annotation: Why Foodborne Disease Surveillance Is Critical to the Safety of Our Food Supply

In the United States, surveillance for foodborne disease traditionally has been conducted for three purposes: to understand the causes of foodborne disease, to allow for the development of appropriate control measures, and to detect trends in the occurrence of foodborne disease.[1] The history of foodborne disease surveillance is rich with tales of outbreaks and investigations, such as the successful control of milkborne diseases through pasteurization and the recent emergence of *Salmonella enteritidis* in eggs and *Escherichia coli* O157:H7 in ground meat products.

**Editor's Note.** See related article by Tilden et al. (p 1142) in this issue.

In recent years, the epidemiology of foodborne disease has been changing because of the increased consumption of raw or minimally processed foods, the consumption of foods out of the home, the globalization of our food supply, and the mass production and distribution of ready-to-eat foods.[1] It is in the context of these changes in diet and industry that pathogens such as *E. coli* O157:H7 have emerged as public health problems.

In this issue of the Journal, Tilden and colleagues demonstrate how an outbreak of *E. coli* O157:H7 occurred in a traditional food-processing operation where everything seemed to be working in accordance with federal regulations and industry-developed good manufacturing practices.[2] A new hazard—the ability of *E. coli* O157:H7 to survive fermentation and drying—was identified. In this regard, the authors' investigation represents the best of foodborne disease surveillance: a hazard was identified, leading to the development of new control measures. It also illustrates why foodborne disease surveillance is critical to maintaining the safety of our food supply.

As manufacturing processes and distribution systems have grown in complexity, so have the outbreaks of foodborne disease associated with them. The low-level contamination of products, demonstrated by Tilden et al. and others, has resulted in the increased occurrence of widely dispersed outbreaks of disease in which individual cases appear as apparently sporadic infections.[1] To recognize these outbreaks requires the efforts of both public health laboratories and the acute disease epidemiologists who must work with them virtually hand-in-hand.

Public health surveillance of foodborne disease is critical to the performance of food safety systems that are based on hazard analysis and critical control point plans. Surveillance is required to identify new hazards, as in the case of *E. coli* O157:H7 in dry fermented salami. It also provides the ultimate feedback on the efficacy of the standard industry safety plans. In the recent nationwide outbreak of *S. enteritidis* infections associated with Schwan's ice cream, the manufacturer's safety plan did not identify transportation as a potential hazard. The outbreak was a result of low-level contamination of pasteurized ice cream "pre-mix" that was transported in the same tanker trailers that also transported raw eggs.[3] Low levels of contamination of this sort that can cause widespread outbreaks of human illness have made humans the "ultimate bioassay" for bacterial pathogens in our food supply.[1] Microbiologic testing of products is not sufficiently sensitive to prevent the occurrence of outbreaks, nor is it necessarily sensitive enough to reliably identify contaminated products during the course of an outbreak.

Epidemiologic methods of foodborne disease surveillance are needed to detect outbreaks, identify their cause, and provide the final assessment of the effectiveness of control measures. To accomplish this, public health officials need resources and the cooperation of health care providers. Surveillance systems for *E. coli* O157:H7, salmonella, and other bacterial enteropathogens begin with physicians ordering stool cultures on patients with diarrhea. Because most foodborne diseases have nonspecific clinical presentations, identification of the organism by culture is necessary to confirm the diagnosis. This is the crucial first step in any outbreak investigation. Culture should be ordered, in particular, for patients with diarrhea and fever or bloody diarrhea, or for patients thought to be part of an outbreak. Although physicians may not always perceive a direct benefit to the patient in ordering a culture in these cases, there may be a community benefit: detecting a foodborne outbreak. Outbreaks typically are identified through individuals. It also should be noted that any individual with watery diarrhea would benefit from oral rehydration therapy.[4] Thus, evaluating a patient with diarrhea is the first step in individual and community interventions.

Public health surveillance for foodborne disease requires resources for laboratories and epidemiologists and the active participation of health care providers. Without these resources, under modern conditions of food manufacture and supply, the role of foodborne disease surveillance in maintaining the safety of our food supply will be greatly diminished. □

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## References

1. Hedberg CW, MacDonald KL, Osterholm MT. Changing epidemiology of food-borne disease: a Minnesota perspective. *Clin Infect Dis.* 1994;18:671–682.
2. Tilden J Jr, Young W, McNamara A-M, et al. A new route of transmission for *Escherichia coli:* infection from dry fermented salami. *Am J Public Health.* 1996;86:1142–1145.
3. Hennessy TW, Hedberg CW, Slutsker L, et al. A national outbreak of *Salmonella enteritidis* infections from ice cream. *N Engl J Med.* 1996;334:1281–1286.
4. Goepp JG, Kate SA. Oral rehydration therapy. *Am Fam Physician.* 1993;47:843–848.

# Annotation: Needle Exchange Programs and the Law—Time for a Change

In his social history of venereal disease, *No Magic Bullet,* Allan M. Brandt describes the controversy in the US military about preventing venereal disease among soldiers during World War I.[1] Should there be a disease prevention effort that recognized that many young American men would succumb to the charms of French prostitutes, or should there be a more punitive approach to discourage sexual contact? Unlike the New Zealand Expeditionary forces, which gave condoms to their soldiers, the United States decided to give American soldiers after-the-fact, and largely ineffective, chemical prophylaxis. American soldiers also were subject to court martial if they contracted a venereal disease. These measures failed. More than 383 000 soldiers were diagnosed with venereal diseases between April 1917 and December 1919 and lost seven million days of active duty. Only influenza, which struck in an epidemic, was a more common illness among servicemen.

This grim lesson was lost on Americans back home. Campaigns against syphilis continued to emphasize abstinence. By the 1930s, almost one in ten Americans was infected with syphilis.

During World War II, however, the American armed forces took a more realistic approach and distributed 50 million condoms each month during the war. The military's new motto—"If you can't say no, take a pro"—recognized that abstinence is the best way to prevent venereal disease, but for those who don't

**Editor's Note.** See related article by Burris et al. (p 1161) in this issue.